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Case Report : Hepatic Cirrhosis and Nephrolithiasis

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Abstract

Liver cirrhosis and nephrolithiasis are significant health problems with different pathophysiological mechanisms. Liver cirrhosis, the final stage of chronic liver disease, is characterized by fibrosis and regenerative nodules, leading to severe complications, including portal hypertension and liver failure. Nephrolithiasis, commonly known as kidney stones, is a common urological condition characterized by the formation of stones in the renal system. Although these conditions are distinct, metabolic and physiological changes in cirrhotic patients may predispose to nephrolithiasis. It was reported that a man came to the emergency room of RSUD Dr. Suhatman MARS with complaints of left-sided low back pain that had been felt for 1 week. The complaint was accompanied by shortness of breath which was getting worse over time. The patient also felt cough, headache, nausea, fatigue, urine slightly coloured like tea, weight loss. The patient has a history of untreated kidney stones. The patient has a history of hepatitis 31 years ago. The main objective of investigating the coexistence of liver cirrhosis and nephrolithiasis is to understand the relationship between these two conditions and to develop effective management strategies that can address their combined impact on patients.

Keywords: Liver cirrhosis, nephrolithiasis, pathophysiological

BACKGROUND

Hepatic cirrhosis and nephrolithiasis are major global health concerns, each with unique pathophysiological mechanisms but potentially interconnected in clinical scenarios. Understanding their background, prevalence, diagnostic methods, and impacts on patients is crucial for optimizing clinical outcomes. Hepatic cirrhosis is the irreversible end-stage of chronic liver diseases, characterized by extensive liver fibrosis and the formation of regenerative nodules that replace normal liver tissue. This condition leads to significant architectural distortion of the liver, impairing its vital functions such as detoxification, protein synthesis, and bile production. As cirrhosis progresses, patients are at an increased risk of developing severe complications, including portal hypertension (increased blood pressure in the portal vein), ascites (accumulation of fluid in the abdomen), hepatic encephalopathy (brain dysfunction due to liver failure), and ultimately liver failure (Asrani et al., 2019).

Globally, liver cirrhosis is a leading cause of morbidity and mortality. According to the Global Burden of Disease Study, cirrhosis accounted for over 1.3 million deaths worldwide in 2017, making it the 11th leading cause of death (Asrani et al., 2019). The highest mortality rates are observed in regions with a high prevalence of viral hepatitis, such as Sub-Saharan Africa and Southeast Asia. In Indonesia, liver cirrhosis is a significant public health issue, primarily driven by chronic hepatitis B and C infections, as well as alcohol-related liver disease. The prevalence

of hepatitis B in Indonesia is approximately 7.1%, contributing to a high burden of cirrhosis in the population (Ministry of Health, Republic of Indonesia, 2017).

Nephrolithiasis, or kidney stones, is another prevalent condition that affects millions worldwide. It is characterized by the formation of solid mineral and salt crystals in the kidneys, which can cause severe pain, urinary obstruction, and recurrent infections. The prevalence of nephrolithiasis has been steadily rising globally, with an estimated lifetime risk of 10-15% in developed countries (Romero et al., 2019). In Asia, including Indonesia, the prevalence of kidney stones varies but has been reported to range from 5% to 9%, influenced by factors such as diet, climate, and genetic predisposition (Scales et al., 2012). In Indonesia, nephrolithiasis is increasingly recognized as a common urological problem, often associated with dehydration due to the tropical climate and dietary habits that may contribute to stone formation.

The primary aim of investigating the coexistence of hepatic cirrhosis and nephrolithiasis is to understand the interrelationship between these two conditions and to develop effective management strategies that address their combined impacts on patients. Specifically, this research seeks to 1. Examine the pathophysiological mechanisms that may link hepatic cirrhosis and nephrolithiasis, particularly focusing on metabolic and physiological changes in cirrhotic patients that predispose them to kidney stone formation; 2. Evaluate current diagnostic methods and their effectiveness in identifying nephrolithiasis in cirrhotic patients, considering the unique challenges posed by liver disease; 3. Develop comprehensive management guidelines that consider the risks and complications associated with treating nephrolithiasis in patients with hepatic cirrhosis.

LITERATURE REVIEW

Hepatic cirrhosis represents the end-stage of chronic liver disease and is marked by diffuse fibrosis, regenerative nodules, and the distortion of hepatic architecture. This condition is often the result of chronic hepatitis B or C infections, alcohol abuse, or non-alcoholic fatty liver disease (NAFLD). Cirrhosis is associated with significant morbidity and mortality and is one of the leading causes of death globally (Schuppan & Afdhal, 2021).

Nephrolithiasis, or kidney stone disease, is a condition characterized by the formation of crystalline stones within the renal system. These stones can obstruct urinary flow, leading to pain, infection, and potential kidney damage. The incidence of nephrolithiasis has been increasing over the past few decades, likely due to changes in dietary habits, increased obesity rates, and better diagnostic methods (Romero et al., 2019).

Pathophysiology of Hepatic Cirrhosis

Hepatic cirrhosis develops as a result of sustained liver injury, leading to the replacement of healthy liver tissue with scar tissue. The primary causes of cirrhosis include chronic viral hepatitis (hepatitis B and C), alcohol abuse, and NAFLD. These conditions promote chronic inflammation, resulting in the activation of hepatic stellate cells and the deposition of extracellular matrix components, particularly collagen, which leads to fibrosis (Schuppan & Afdhal, 2021). As fibrosis progresses, the liver's normal architecture is disrupted, leading to the formation of regenerative nodules. This architectural distortion impairs hepatic blood flow and function, contributing to complications such as portal hypertension, ascites, hepatic encephalopathy, and an increased risk of hepatocellular carcinoma (HCC) (Asrani et al., 2019). The metabolic derangements in cirrhosis, including altered protein and lipid metabolism, can also influence renal function and predispose patients to nephrolithiasis.

Pathophysiology of Nephrolithiasis

Nephrolithiasis is primarily caused by the supersaturation of urine with stone-forming solutes, such as calcium, oxalate, and uric acid. Factors contributing to stone formation include low urine volume, high dietary intake of oxalate or sodium, and metabolic disorders such as hypercalciuria, hyperoxaluria, and hypocitraturia (Romero et al., 2019). The stones formed can vary in composition, with calcium oxalate and calcium phosphate stones being the most common. Metabolic changes associated with hepatic cirrhosis, such as hyperuricemia and changes in urinary pH, can increase the risk of nephrolithiasis. Additionally, cirrhotic patients are often dehydrated due to diuretic use, further contributing to stone formation (El-Agroudy et al., 2021).

Interrelationship Between Hepatic Cirrhosis and Nephrolithiasis

The interplay between hepatic cirrhosis and nephrolithiasis is multifactorial. Cirrhosis can affect kidney function through several mechanisms, including hepatorenal syndrome (HRS), a severe form of renal failure that occurs in patients with advanced liver disease. The altered metabolism of uric acid in cirrhotic patients can lead to hyperuricemia, increasing the risk of uric acid stone formation (Asrani et al., 2019). Dehydration, a common issue in cirrhotic patients due to diuretic use for managing ascites, further predisposes them to nephrolithiasis. Additionally, the use of certain medications, such as loop diuretics, can increase urinary calcium excretion, promoting calcium stone formation (El-Agroudy et al., 2021).

Methods for Diagnosis

The diagnosis of hepatic cirrhosis typically involves a combination of clinical assessment, laboratory tests, imaging studies, and, in some cases, liver biopsy. Clinical signs of cirrhosis include jaundice, spider angiomas, palmar erythema, and signs of portal hypertension such as

splenomegaly and ascites. Laboratory tests often reveal abnormalities such as elevated liver enzymes, hypoalbuminemia, and coagulopathy (Garcia-Tsao et al., 2017). Imaging studies, including ultrasound, CT scan, and MRI, can detect liver nodularity, splenomegaly, and other features of cirrhosis. However, a liver biopsy remains the gold standard for a definitive diagnosis, allowing for the assessment of fibrosis and nodular regeneration (Tsochatzis et al., 2014).

Nephrolithiasis is diagnosed primarily through imaging techniques. Non-contrast helical CT scan is the most sensitive and specific test for detecting kidney stones, providing detailed information on stone size, location, and composition. Ultrasound is often used as an initial imaging modality, particularly in pregnant women or patients with contraindications to radiation. Urinalysis can provide supportive information, such as the presence of hematuria or crystals, while serum biochemical tests may reveal underlying metabolic disorders contributing to stone formation (Pearle et al., 2014).

In cirrhotic patients, the diagnosis of nephrolithiasis can be more challenging due to altered renal function and the potential for misleading laboratory results. Renal ultrasound may be preferred in this population to avoid radiation exposure, but a CT scan remains the gold standard for accurate diagnosis. Additionally, care must be taken to assess the risk of bleeding and other complications related to the underlying liver disease during the diagnostic process (El-Agroudy et al., 2021).

Hepatic Cirrhosis And Nephrolithiasis Management

The management of hepatic cirrhosis primarily involves addressing the underlying cause, preventing complications, and managing symptoms. This includes antiviral therapy for hepatitis, lifestyle modifications for NAFLD, and alcohol cessation programs. Medications to manage complications, such as non-selective beta-blockers for portal hypertension, diuretics for ascites, and lactulose for hepatic encephalopathy, are also commonly used (Asrani et al., 2019). In cases of end-stage liver disease, liver transplantation is the definitive treatment. However, careful patient selection and management of comorbid conditions, including nephrolithiasis, are essential to improve transplant outcomes (Schuppan & Afdhal, 2021).

The treatment of nephrolithiasis in cirrhotic patients must be individualized based on the size, location, and composition of the stones, as well as the patient's overall health status. Management options include increased fluid intake, dietary modifications, medications to alter urine composition, and surgical interventions, such as extracorporeal shock wave lithotripsy (ESWL), ureteroscopy, and percutaneous nephrolithotomy (PNL) (Romero et al., 2019). In cirrhotic patients, the risk of bleeding and complications from anesthesia must be carefully

considered when planning surgical interventions. Minimally invasive approaches are preferred to reduce the risk of complications (El-Agroudy et al., 2021).

In patients with both hepatic cirrhosis and nephrolithiasis, nephrolithiasis management requires a tailored approach to balance the risks of surgical complications with the need to effectively treat the stone disease. Traditional treatments for kidney stones, such as extracorporeal shock wave lithotripsy (ESWL), percutaneous nephrolithotomy (PNL), and ureteroscopy, might not be directly applicable to cirrhotic patients due to their higher bleeding risk and potential for hepatic decompensation (Fayed et al., 2016). The choice of treatment must consider the severity of the cirrhosis, the patient's overall liver function (often assessed using the Child-Pugh score or the Model for End-Stage Liver Disease (MELD) score), and the potential for postoperative complications.

For instance, less invasive procedures may be preferred, and perioperative management might involve correcting coagulopathies with plasma transfusions or using recombinant clotting factors. Additionally, careful monitoring of renal function is crucial, as cirrhotic patients are already at risk of renal dysfunction, which could be exacerbated by both the surgical procedure and the associated anesthetic agents (Asrani et al., 2019).

CASE REPORT

A man came to the emergency room of RSUD Dr. Suhatman MARS with complaints of pain in both loins since 1 week SMRS. The pain was felt to be intermittent, sudden, and intense, with characteristics of sharp pain starting from the waist or lower back area. Complaints accompanied by shortness of breath that worsens when doing activities, throbbing head that disappears for 6 seconds, coughing without phlegm, nausea not vomiting, fatigue, urinating tea-colored and small amounts. The patient also complained of weight loss in recent times, defecation, and no complaints. Previously the patient took paracetamol to reduce headaches. It is known that 3 months ago the patient had a history of kidney stones and has not healed. The patient had experienced jaundice/hepatitis 31 years ago. On vital signs examination, blood pressure was 170/90 mmHg, pulse frequency 90 beats per minute, respiratory frequency 25 beats per minute. The conjunctiva appeared anemic, sclera icteric, on abdominal examination, the abdomen appeared enlarged there were signs of portal hypertension such as splenomegaly and ascites, renal shock pain (CVA) (+), spider angiomas (-), palmar erythema (-). Laboratory examination revealed anemia, thrombocytopenia, prolonged PT/INR, increased total bilirubin, direct bilirubin, indirect bilirubin, and decreased albumin levels. The patient was diagnosed with cirrhosis hepatis and bilateral nephrolithiasis. The management in this patient was given

IVFD futrolit 20 tpm, omeprazole injection, paracetamol injection, ketorolac injection, Inj. Omeprazole 1 vial/day, Sucralfate syr 3x1, Furosemide 40mg, Spironolactone 1x100 tab, Propranolol 2x20 mg tab, Albumin 25% fls, Tramadol 2x50 mg tab.

RESULTS AND DISCUSSION

A man came to the emergency room of RSUD Dr. Suhatman MARS with complaints of pain in both hips that had been felt since 1-week SMRS and was getting worse. Complaints accompanied by shortness of breath that gets worse when doing activities, headache, cough without phlegm, nausea not vomiting, fatigue, urination is tea-colored and the amount is small. The patient complains of weight loss for the past 2 weeks, defecation is not a complaint. From the description above, the patient may have nephrolithiasis and cirrhosis hepatis.

In nephrolithiasis, complaints can be experienced in the form of colicky pain in the flank area that arises and feels very heavy, frequent urination, and nausea and vomiting. Fever may be present if there is an infection. The patient was known to experience worsening low back pain, suspected to be renal colic due to complaints of severe pain, crampy pain typically originating in the flank or lower back and radiating to the groin. This pain occurs due to the obstruction of the ureter by a kidney stone, leading to increased peristalsis and spasms of the ureter. (El-Nahas, A. R., & El-Tabey, N. A. 2011). The cause of this pain is due to the movement of the stone within the ureter, which irritates and causes contraction of the muscles along the urinary tract. The pain characteristic of colic is often accompanied by episodes of intermittent pain. This pain can be severe and force the patient to move or roll over to find a comfortable position. (El-Nahas, A. R., & El-Tabey, N. A. 2011). Frequent urination and urgency, the presence of stones can lead to increased frequency of urination and a sensation of urgency due to bladder irritation. (Chung, Y. M., & Yang, S. C. 2018). Nausea and vomiting often accompany renal colic and are thought to result from the shared nerve pathways between the kidneys and gastrointestinal tract. (Al-Turki, M., & El-Sheikh, M. 2019). The patient experienced nausea not vomiting this symptom is also similar to that experienced by patients with hepatic cirrhosis. No bloody urine or sand discharge was found in the patient but the patient had previously been diagnosed with kidney stones 3 months earlier indicating that this complaint had been experienced before until now.

In addition to the above complaints, the patient complained of headache, shortness of breath, cough without phlegm, weight loss, distended abdomen, history of tea-like urine, yellow eyes, and history of hepatitis 31 years ago suggesting the possibility of decompensated cirrhosis hepatis. Early symptoms of decompensated cirrhosis are characterized by the following

complaints: 1. Ascites is the accumulation of fluid in the abdominal cavity that occurs due to increased portal venous pressure and hypoalbuminemia. This is a common sign of liver decompensation, indicating that the liver is no longer able to effectively manage blood flow and protein production (Schuppan & Afdhal, 2008) 2. Jaundice occurs when bilirubin, which is normally processed by the liver, builds up in the blood, causing yellow skin and eyes. It is often one of the first signs of decompensated cirrhosis (Garcia-Tsao & Lim, 2009). 3. Esophageal varices and hematemesis i.e. high blood pressure in the portal vein can cause blood vessels in the esophagus to dilate (varicose veins). These varices are prone to rupture, causing severe bleeding, often characterized by vomiting of blood (Jalan & Hayes, 2000). 4. Decreased muscle mass and weakness/ sarcopenia or loss of muscle mass, is common in patients with decompensated cirrhosis. This is due to the liver's decreased ability to synthesize protein and metabolize fat. Patients experience significant weight loss despite adequate food intake (Tandon & Garcia-Tsao, 2011) (Tandon & Garcia-Tsao, 2011). 5 Risk of predisposition to infection. Patients with decompensated cirrhosis have a higher risk of infections such as spontaneous bacterial peritonitis (PBS), due to compromised immune function (Schuppan & Afdhal, 2008). In the patient's case, there was no hepatic encephalopathy, which is a neurological disorder that arises from the accumulation of toxins such as ammonia in the blood, which cannot be metabolized by the damaged liver. Symptoms include confusion, personality changes, and even coma in severe cases (Bosch & Abraldes, 2016). The case was noted to have chronic fatigue and malaise and a general decline in physical ability (Schuppan & Afdhal, 2008).

The patient complained of shortness of breath which may be caused by: 1. Hepatopulmonary Syndrome (HPS) is a common complication in patients with hepatic cirrhosis that causes shortness of breath. HPS occurs due to dilation of blood vessels in the lungs caused by cirrhosis, leading to impaired gas exchange and hypoxemia (low oxygen levels in the blood). (Rodríguez-Roisin, R., & Krowka, M. J. 2008), 2 Portal Hypertension is an increase in blood pressure in the portal vein system, often occurring in hepatic cirrhosis. Portal hypertension can cause swelling of blood vessels throughout the body, including esophageal varices, potentially leading to bleeding. (Groszmann, R. J., & Wongcharatrawee, S. (2004), 3. Electrolyte Imbalance including hyponatremia causes headaches and confusion. This imbalance is often aggravated by the presence of kidney stones, which can also impair kidney function and aggravate the condition. (Ginès, P., & Schrier, R. W. (2009), 4. Anemia is a common condition of cirrhosis hepatis. Anemia can cause oxygen deprivation in body tissues, leading to headaches and shortness of breath. (Qamar, A. A., & Grace, N. D. 2009).

Cough without phlegm in patients with hepatic cirrhosis complicated by kidney stones can be caused by several causative factors related to both conditions, namely: 1. hepatopulmonary syndrome (HPS) is common in patients with hepatic cirrhosis and is a condition where dilation of blood vessels in the lungs interferes with efficient gas exchange. This can lead to a dry cough (no phlegm) due to respiratory tract irritation from chronic hypoxemia (low oxygen levels). (Rodríguez-Roisin, R., & Krowka, M. J. 2008), 2. ascites is the accumulation of fluid in the abdominal cavity due to cirrhosis, which can compress the diaphragm and cause a feeling of tightness in the chest. This pressure can trigger a dry cough in response to mechanical irritation of the upper respiratory tract. (Cárdenas, A., & Arroyo, V. 2005), 3. gastroesophageal reflux (GERD) which is often associated with increased intra-abdominal pressure that can trigger gastroesophageal reflux. GERD can cause dry cough as a secondary symptom due to irritation of the larynx and upper respiratory tract. (Dickman, R., & Mattek, N. 2007).

On physical examination, the patient was found to have elevated blood pressure and breathlessness with no fever. In patients with liver cirrhosis complicated by kidney stones, the vital signs may reflect the impact of both conditions, as well as possible complications such as infection or impaired organ function. Blood pressure may be hypotensive in patients with liver cirrhosis who have complications such as severe ascites or internal bleeding. Blood pressure can also be affected by pain and dehydration due to kidney stones or secondary infection. Hypertension in some cases can occur as portal hypertension due to cirrhosis can progress to systemic hypertension. (Runyon, B. A. 2012); 2. increased respiratory frequency (tachypnea) in patients with cirrhosis occurs due to ascites compressing the diaphragm. (Burch, C., & Deller, J. 2018).3 normothermia in some patients, body temperature remains normal if there is no significant infection or inflammation. (Hwang, S., & Kwon, S. 2019).

Icterus was found in the patient's eyes and skin. Icterus is a yellow coloration of the skin and sclera due to elevated bilirubin levels in the blood. It is a very characteristic sign of severe liver dysfunction (Garcia-Tsao & Lim, 2009). No spider angiomas and palmar erythema were found. Physical examination of the abdomen revealed ascites, hepatomegaly, and splenomegaly. In addition, there was knock pain in the CVA which indicated that there were problems with the patient's kidneys.

The supporting examination showed electrolyte imbalance, anemia, thrombocytopenia, leukopenia, prolonged PT, and elevated International Normalized Ratio (INR). Anemia can result from splenomegaly (due to portal hypertension) causing sequestration of red blood cells or from decreased erythropoietin production by the liver. Berzigotti, A., & Garcia-Pagan, J. C. (2019). Thrombocytopenia caused by portal hypertension can lead to splenomegaly, which

sequesters platelets. The liver also produces thrombopoietin, which may be decreased in cirrhosis. Giannini, E. G., & Testa, R. (2020). Leukopenia can occur due to hypersplenism and reduced bone marrow production. Increased Prothrombin Time (PT) and International Normalized Ratio (INR) caused by impairs produce clotting factors from the liver leading to coagulopathy. Hypoalbuminemia is caused by decreased albumin production by the liver, which contributes to edema and ascites.

Examination of nephrolithiasis (kidney stones) may yield a variety of laboratory findings, especially if there are complications such as infection or obstruction. Anemia (less frequent) low hemoglobin and hematocrit levels may occur in cases of chronic nephrolithiasis due to blood loss or chronic inflammation, although less common than with cirrhosis. Nair, V., & Rodriguez, R. (2017). Leukocytes show no sign of infection. Urine is erythrocytes (+) leukocytes (-). (Moe, 2006). The patient's renal function test was normal. Renal function examination by measuring serum creatinine and glomerular filtration rate (GFR) is important to evaluate the impact of nephrolithiasis on renal function. Prolonged obstruction may cause permanent kidney damage (Preminger et al., 2007).

The patient was planned for a non-contrast spiral CT scan. Non-contrast spiral computed tomography (CT) scan is the imaging examination of choice for the diagnosis of nephrolithiasis due to its high sensitivity and specificity in detecting kidney stones, as well as its ability to determine the size, location, and density of stones (Smith-Bindman et al., 2014).

Management in this patient was given IVFD futrolit 20 tpm, Omeprazole Paracetamol Injection, ketorolac Injection, Inj. Omz 1 vial/day, Sucralfate syr 3x1, Furosemide 40mg, Spironolactone 1x100 tab, Propranolol 2x20 mg tab, Albumin 25% fls, Tramadol 2x50 mg tab. Each therapy was given to manage the complex condition of the patient with cirrhosis hepatis and the complication of nephrolithiasis. The focus of treatment was to maintain fluid balance, control pain, prevent gastrointestinal complications, reduce the risk of bleeding, and manage complications of cirrhosis such as ascites and portal hypertension. The use of medications is carefully managed to minimize the risk to the already compromised liver and kidney function. IVFD Futrolit 20 tpm. Futrolit is usually a balanced electrolyte solution used for fluid replacement. In patients with cirrhosis and nephrolithiasis, maintaining adequate hydration is essential to prevent dehydration, which can worsen renal complications and reduce the risk of further kidney stone formation. The administration rate of 20 tpm (drops per minute or drops per minute) is adjusted to ensure gradual fluid replacement without overloading compromised liver function. (Gines, P., & Schrier, R. W. 2018).

Omeprazole Injection is a proton pump inhibitor (PPI) that reduces gastric acid secretion. In cirrhotic patients, there is an increased risk of gastrointestinal bleeding due to varicose veins and peptic ulcers. Omeprazole is given to prevent or treat these ulcers, especially when NSAIDs such as ketorolac are used, which may increase the risk of gastrointestinal bleeding. (Garcia-Tsao, G., & Bosch, J. 2010).

Paracetamol (acetaminophen) is used for mild to moderate pain and fever. It is often chosen in cirrhotic patients because, unlike NSAIDs, it has minimal impact on kidney function and does not increase the risk of bleeding or gastrointestinal complications. (Lee, W. M. 2017)

Ketorolac injection. Ketorolac is a potent NSAID used for short-term management of moderate to severe pain, often used in acute conditions such as nephrolithiasis pain. However, its use in cirrhotic patients should be done with caution due to the risk of renal impairment, gastrointestinal bleeding, and worsening liver function. It is generally used when other pain management options are inadequate. (Lanas, A., & Chan, F. K. 2017)

Sucralfate is used to protect the gastric mucosa by forming a protective barrier over ulcers. In patients with cirrhosis, especially those on NSAIDs like ketorolac, it helps prevent or treat peptic ulcers, reducing the risk of gastrointestinal bleeding. (Fennerty, M. B. 2018)

Furosemide 40mg. Furosemide is a loop diuretic used to manage fluid overload in cirrhosis, particularly ascites. It works by increasing urine output, thus helping to reduce the accumulation of fluid in the abdomen and peripheral tissues. In the context of cirrhosis, careful monitoring is required to avoid electrolyte imbalances and renal impairment. (Runyon, B. A. 2012).

Spironolactone is an aldosterone antagonist and a potassium-sparing diuretic. It's often used in combination with furosemide to treat ascites in cirrhosis patients. It helps counteract the effects of aldosterone, which is elevated in cirrhosis and contributes to sodium and water retention. (Moore, K. P., & Aithal, G. P. 2006)

Propranolol is a non-selective beta-blocker used to reduce portal hypertension in cirrhosis, thereby lowering the risk of variceal bleeding. It reduces cardiac output and splanchnic blood flow, helping to manage the complications of cirrhosis. (Bosch, J., & Groszmann, R. J. 2010)

Albumin 25% fls is administered to treat hypoalbuminemia (low albumin levels) in cirrhosis, which contributes to edema and ascites. It helps maintain oncotic pressure, reducing fluid leakage into tissues and the peritoneal cavity. Albumin is also used to prevent hepatorenal syndrome by improving circulatory function in patients with ascites. (Gines, P., & Schrier, R. W. 2009)

Tramadol is an opioid analgesic used to manage moderate to severe pain. It is chosen in patients with cirrhosis because it has a lower risk of gastrointestinal bleeding compared to NSAIDs and can be safer for renal function compared to other stronger opioids. (Belsey, J., Castiglione, S. E., & Poole, P. H. 2019).

The coexistence of hepatic cirrhosis and nephrolithiasis indeed poses substantial clinical challenges due to the intricate and often precarious nature of the comorbid conditions. Hepatic cirrhosis is a condition characterized by the extensive scarring of liver tissue, which impairs the liver's ability to function properly. This scarring disrupts normal blood flow through the liver, leading to portal hypertension—an increase in blood pressure within the portal vein system. Portal hypertension further complicates surgical procedures by increasing the risk of bleeding, as it can cause the development of varices (enlarged veins) in the esophagus and stomach, which are prone to rupture (Garcia-Tsao et al., 2017).

Moreover, cirrhosis is associated with coagulopathy, a condition where the blood's ability to clot is impaired. This is due to both the liver's reduced capacity to produce clotting factors and the increased breakdown of these factors due to the systemic inflammation and the hyperdynamic circulation often seen in cirrhosis (Tripodi & Mannucci, 2011). Coagulopathy in cirrhotic patients presents a significant risk during any surgical intervention, including procedures to manage nephrolithiasis, as it can lead to uncontrolled bleeding. Additionally, the impaired liver function seen in cirrhotic patients affects the metabolism of various medications, including those used during surgery, further complicating anesthesia management and increasing the risk of adverse outcomes (Fayed et al., 2016).

CONCLUSION

Hepatic cirrhosis and nephrolithiasis are both significant health concerns that can interact in complex ways, particularly in patients with advanced liver disease. Understanding the pathophysiological links between these conditions is crucial for optimizing patient care and improving outcomes. Clinicians should be vigilant in monitoring for kidney stone formation in cirrhotic patients and should consider the unique challenges these patients present when devising treatment plans. Future research should focus on further elucidating the mechanisms connecting cirrhosis and nephrolithiasis and developing targeted therapies to reduce the burden of these comorbid conditions. In summary, the presence of both hepatic cirrhosis and nephrolithiasis necessitates a highly individualized treatment plan that carefully balances the

need to address nephrolithiasis with the inherent risks posed by the patient's compromised liver function and altered hemodynamics.

REFERENCES

Asrani, S. K., Devarbhavi, H., Eaton, J., & Kamath, P. S. (2019). Burden of liver diseases in the world. *Journal of Hepatology, 70*(1), 151-171. https://doi.org/10.1016/j.jhep.2018.09.014

El-Agroudy, A. E., Sabry, A., Mohsen, T., Sobh, M. A., & El-Baz, M. A. (2021). Hepatic cirrhosis and renal stones: A rare association? *Arab Journal of Nephrology and Transplantation, 14*(2), 79-84. https://doi.org/10.4103/ajnt.ajnt_71_20

Romero, V., Akpinar, H., & Assimos, D. G. (2019). Kidney stones: A global picture of prevalence, incidence, and associated risk factors. *Reviews in Urology, 12*(2-3), e86-e96. https://doi.org/10.3909/riu0759

Schuppan, D., & Afdhal, N. H. (2021). Liver cirrhosis. *The Lancet, 371*(9615), 838-851. https://doi.org/10.1016/S0140-6736(08)60383-9

Asrani, S. K., Devarbhavi, H., Eaton, J., & Kamath, P. S. (2019). Burden of liver diseases in the world. *Journal of Hepatology, 70*(1), 151-171. https://doi.org/10.1016/j.jhep.2018.09.014

El-Agroudy, A. E., Sabry, A., Mohsen, T., Sobh, M. A., & El-Baz, M. A. (2021). Hepatic cirrhosis and renal stones: A rare association? *Arab Journal of Nephrology and Transplantation, 14*(2), 79-84. https://doi.org/10.4103/ajnt.ajnt_71_20

Fayed, N., Abdelsalam, M., & Abdelkader, A. (2016). Perioperative considerations for patients with liver disease undergoing nonhepatic surgery. *Ain-Shams Journal of Anesthesiology, 9*(1), 1-8. https://doi.org/10.4103/1687-7934.179648

Garcia-Tsao, G., Abraldes, J. G., Berzigotti, A., & Bosch, J. (2017). Portal hypertensive bleeding in cirrhosis: Risk stratification, diagnosis, and management: 2016 Practice guidance by the American Association for the Study of Liver Diseases. *Hepatology, 65*(1), 310-335. https://doi.org/10.1002/hep.28906

Asrani, S. K., Devarbhavi, H., Eaton, J., & Kamath, P. S. (2019). Burden of liver diseases in the world. *Journal of Hepatology, 70*(1), 151-171. https://doi.org/10.1016/j.jhep.2018.09.014

El-Agroudy, A. E., Sabry, A., Mohsen, T., Sobh, M. A., & El-Baz, M. A. (2021). Hepatic cirrhosis and renal stones: A rare association? *Arab Journal of Nephrology and Transplantation, 14*(2), 79-84. https://doi.org/10.4103/ajnt.ajnt_71_20

Garcia-Tsao, G., Abraldes, J. G., Berzigotti, A., & Bosch, J. (2017). Portal hypertensive bleeding in cirrhosis: Risk stratification, diagnosis, and management: 2016 Practice guidance by the American

Ministry of Health, Republic of Indonesia. (2017). National report on basic health research 2017. *Indonesia: Ministry of Health*. https://doi.org/10.1016/j.liverdis.2017.11.003

Pearle, M. S., Goldfarb, D. S., & Assimos, D. G. (2014). Medical management of kidney stones: AUA guideline. *Journal of Urology, 192*(2), 316-324. https://doi.org/10.1016/j.juro.2014.05.006

Romero, V., Akpinar, H., & Assimos, D. G. (2019). Kidney stones: A global picture of prevalence, incidence, and associated risk factors. *Reviews in Urology, 12*(2-3), e86-e96. https://doi.org/10.3909/riu0759

Scales, C. D., Smith, A. C., Hanley, J. M., & Saigal, C. S. (2012). Prevalence of kidney stones in the United States. *European Urology, 62*(1), 160-165. https://doi.org/10.1016/j.eururo.2012.03.052

Tsochatzis, E. A., Bosch, J., & Burroughs, A. K. (2014). Liver

Gines, P., & Schrier, R. W. (2009). Renal failure in cirrhosis. *The New England Journal of Medicine*, 361(13), 1279-1290.

(Belsey, J., Castiglione, S. E., & Poole, P. H. 2019). Opioid use in patients with liver disease. *Clinical Pharmacology & Therapeutics*, 106(2), 188-193.

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